Agent Orange Investigative Report Series, No. 14

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INVESTIGATION INTO THE ENVIRONMENTAL FATE OF TCDD/DIOXIN

Compensation Service

Department of Veterans Affairs

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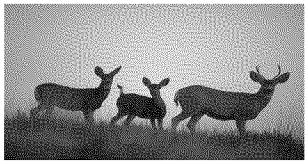

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March 4, 2014
Mr. Michael D. Pharr
Contract Officer's Representative
Compensation Service
Department of Veterans Affairs
810 Vermont Ave., NW
Washington, DC 20420

Dear Mr. Pharr,

Please find attached to this letter the Final Report: **Investigation into the Environmental Fate of TCDD/Dioxin.** This report is the fourteenth of many reports that will be prepared in fulfillment of Contract VA-101-12-C-0006, *Development of an Archival Directory of Agent Orange Documents.* The investigative reports are supported by the archival research. The goal of developing the directory is to search and identify the thousands of documents, reports, and correspondence located within our National Archives and Records Administration and other document repositories that relate to the use of "Tactical Herbicides", including Agent Orange outside of Vietnam. Using documents from the repositories, reports are prepared on topics requested by the Compensation Service.

In the case of this report, the Compensation Service has not prepared an extensive response to questions and claims related to the potential exposure to TCDD, the dioxin contaminant in Agent Orange, by veterans who served within the Continental United States (CONUS) at military installations where the tactical herbicide was tested and evaluated. The focuses of many of these claims are related to the bioavailability of the contaminant 2,3,7,8 -Tetrachlorodibenzo-*p*-dioxin (TCDD) in soils of the test sites. Over a span of four decades thousand of articles have been published on TCDD, making it, not only a chemical of regulatory interest, but one of the most researched molecules worldwide. Unfortunately, some science published about TCDD has been ignored in favor of provocative interpretations and conclusions. This is especially true of the discussions of it environmental fate, which by necessity has been the most difficult to acquire and frequently the most difficult to interpret.

There are three major sources for human exposure related to environmental studies of TCDD: 1) It entered the environment from the improper handling of industrial wastes from the manufacture of chlorophenolic products; 2) It entered the atmosphere as a consequence of an industrial accident or combustion sources; or, 3) It entered the environment as a result of the spraying or spillage of herbicides contaminated with TCDD. From a review of the available data, it was concluded that when 2,3,7,8-TCDD entered the environment, it was rapidly bound to soil and organic particles. Its low water solubility and low vapor pressure resulted in its failure to move in the soil profile, while at the same time these properties enhanced its long -term persistence. In humans, handling contaminated soil resulted in negligible contamination since the skin acted as an effective barrier to the uptake of the TCDD.

Sincerely,

Alvin L. Young

Alvin L. Young, PhD,
Prof. of Environmental Toxicology
Colonel, USAF (Retired)

DISCLAIMER FOR VA REPORTS

The conclusions reached in this report are based upon a comprehensive review of the historical records maintained in the publicly available files of the National Archives and Records Administration, and other archival repositories. However, the conclusions reached do not necessarily represent those of the Department of Veterans Affairs or any other Department or Agency of the United States Government.

This report is part of the Agent Orange Investigative Report Series, and should be considered as an amendable or living document. If additional authenticated documents or records are found that address the topic of this report, a re-evaluation of the conclusions may be necessary.

INVESTIGATION INTO THE ENVIRONMENTAL FATE OF TCDD/DIOXIN

EXECUTIVE SUMMARY

There are three major sources for human exposure related to environmental studies of TCDD: 1) It entered the environment from the improper handling of industrial wastes from the manufacturer of chlorophenolic products; 2) It entered the atmosphere as a consequence of an industrial accident or combustion sources; or, 3) It entered the environment as a result of the spraying or spillage of herbicides contaminated with TCDD. The review of the available data essentially involved examining three case studies: the Missouri Dioxin Episode, the Seveso, Italy Dioxin Episode, and the USAF Environmental Studies of Agent Orange. It was concluded that when 2,3,7,8-TCDD entered the environment, it was rapidly bound to soil and organic particles. Its low water solubility and low vapor pressure resulted in its failure to move in the soil profile, while at the same time these properties enhanced its long-term persistence.

In water sources contaminated with TCDD, the residue was found to be bound to the soil particles that comprised the soil sediment; certain aquatic species were contaminated from ingesting the sediment. Animals that came into direct contact with a liquid matrix containing the TCDD were likely poisoned. However, animals that came into contact with "aged" contaminated soil could become contaminated, but the level of contamination was generally insufficient to have adverse effects upon the animals. In humans, handling contaminated soil resulted in negligible contamination since the skin acted as an effective barrier to the uptake of the TCDD.

INTRODUCTION

The concern over the widespread military use of tactical herbicides in the Vietnam War, especially the use of Agent Orange, stemmed primarily from the dioxin (2,3,7,8-tetrachlorodibenzo-*p*-dioxin, TCDD) contaminant in the 2,4,5-T herbicide. Our awareness of its chemistry, toxicity, persistence in biological tissue, and environmental fate now spans almost 40 years. During this span of four decades thousands of articles have been published on TCDD, making it not only a chemical of regulatory interest but one of the

most researched molecules worldwide. Unfortunately, some science published about TCDD has been ignored in favor of provocative interpretations and conclusions. This is especially true of the discussions of its environmental fate, which by necessity has been the most difficult to acquire and frequently the most difficult to interpret.

The IOM VIEWS ON VIETNAM VETERAN EXPOSURES

The results of the Institute of Medicine's comprehensive reviews of occupational, environmental, and veterans' studies conducted over the past 18 years have been provided periodically to the Secretary of Veterans Affairs, together with an extensive list of IOM's findings "regarding the association between specific health problems (illnesses) and exposure to herbicides" [1]. However, the IOM has not provided evidence or findings of the veterans likely levels of exposure to or absorption of herbicides or 2,3,7,8-TCDD. Indeed, the IOM viewed the determination of exposure of US military personnel who served in Vietnam as "perhaps the greatest challenge in the study of health effects associated with herbicides and TCDD." The IOM added the following explanation:

> *Some military personnel stationed in cities or on large bases may have received little or no herbicide exposure, whereas troops who moved through defoliated areas soon after treatment may have been exposed through soil contact, drinking water, or bathing. Reliable estimates of the magnitude and duration of such exposures are not possible in most cases, given the lack of contemporaneous chemical measurements, the lack of a full understanding of the movement and behavior of the defoliants in the environment, and the lack of records of individual behaviors and locations* [1].

The IOM additionally recognized that a focus on aerial spraying as the primary exposure may be misplaced and what is needed is a total exposure assessment that accounts for all sources and routes of exposure [1]. Recognizing the difficulty for Vietnam veterans to document their exposures to Agent Orange and other tactical herbicides, the Congress passed and the government enacted the Agent Orange Act of 1991. For Vietnam veterans an assessment of exposure became a moot issue since the policy of the Department of Veteran Affairs assumed exposure occurred for all Vietnam veterans who had "boots on the ground" or served in the inland waterways in Vietnam. A similar presumption was given for US veterans who served in Korea along the demilitarized zone (DMZ) between 1 April 1968 and 31

August 1971. However, Vietnam-era veterans who have filed claims but did not qualify for the presumption of exposure must provide Compensation Service with documentation of exposure to Agent Orange or the other tactical herbicides while serving in military service. The role of environmental fate should be key to any determination of claims.

A previous report in the Agent Orange Investigative Series has described the significance of environmental fate on the exposure to the four component herbicides found in the tactical herbicides tested and evaluated within the Continental United States (CONUS) for use in Vietnam [2]. This report focuses on the significance of environmental fate on exposure to the contaminant 2,3,7,8-TCDD (dioxin) found in the herbicide 2,4,5-T.

UNDERSTANDING THE TOXICITY OF TCDD

Dioxins can be released into the environment through forest fires, backyard burning of trash, certain industrial activities, residues from past commercial burning of waste, and from certain chlorinated pesticides [3]. The levels of TCDD and other dioxins present in the environment as a result of industrial and municipal activities, and under conditions representing possible misuse, are also known [3]. The health effects associated with dioxins depend on a variety of factors. They include the level of exposure and duration and number exposure, i.e., a situation in which there is an opportunity of accumulating an actual toxic dose of the chemical within the body [3]. The toxicity of TCDD when administered as a dose is known with reasonable accuracy for dozens of laboratory animals under a variety of conditions. Also, much has been learned about the effects of TCDD on humans from industrial accidents [1]. However, exposure to TCDD correctly means a situation of proximity and potential for intake, it does not mean the actual intake or absorption of a dose (in other words, exposure and dose are not equal). These are important concepts to understand as Compensation Serve evaluates claims from Vietnam-era veterans *outside of Vietnam.*

An example: If a veteran claimed exposure to Agent Orange while in service in a location where Agent Orange had been evaluated, but was not involved at the time in the actual loading or spraying of the tactical herbicide, or not in the test area at the time of spraying, he might claim that he was subsequently "exposed" to residues of Agent Orange and TCDD in the area. The likelihood of actually acquiring a "dose" may be negligible because of the environmental fates of both the herbicides and the TCDD.

In order to realistically assess the human risks inherent in the presence of TCDD in the environment, the information from toxicological studies must be coupled with the likelihood and degree of human exposure, and to that end, environmental fate plays a key role in the assessment.

PHYSICAL AND CHEMICAL PROPERTIES OF TCDD

Knowledge of the physical and chemical properties of 2,3,7,8-TCDD is essential to understanding its environmental behavior. The EPA accepted water solubility value for 2,3,7,8-TCDD is 19.3 ± 3.7 parts per trillion (nanograms per liter, ng/L) at 22° C [4]. The accepted vapor pressure value for 2,3,7,8-TCDD is 1.50×10^{-9} mm Hg at 25° C [5]. The organic carbon partition coefficient (K_{oc}) describes the partitioning of contaminants between suspended sediment and the water column. The accepted K_{oc} value for 2,3,7,8-TCDD is 6.6 [6]. These physical and chemical properties of 2,3,7,8-TCDD suggest that the compound is essentially insoluble in water, tightly bound to particulates and the organic matter in soil and sediments, and would be extremely stable under most environmental conditions. Burial in-place or erosion of soil to water bodies would likely be the predominant fate of 2,3,7,8-TCDD adsorbed to soil [7]. However, the low solubility and vapor pressure (1.62×10^{-5} @25°C) of TCDD predicted that its volatilization half-life in the water of lakes and ponds would be ~ 32 days, while the half-life for rivers would be ~16 days [8].

FIELD STUDIES OF 2,3,7,8-TCDD RESIDUES AND HUMAN EXPOSURE

There are three major sources for human exposure related to environmental studies of TCDD: 1) It entered the environment from the improper handling of industrial wastes from the manufacturer of chlorophenolic products; 2) It entered the atmosphere as a consequence of an industrial accident or combustion sources; or, 3) It entered the environment as a result of the spraying or spillage of herbicides contaminated with TCDD.

Soil Contamination from Industrial Wastes

During 1971-1972, the Northeast Pharmaceutical and Chemical Corporation (NEPACCO) near St. Louis, Missouri arranged for the periodic disposal of still-bottom residues from the production of hexachlorophene. One of the final steps in the process of purifying the hexachlorophene was distillation, and the dioxins (especially 2,3,7,8-TCDD) were concentrated in the residues

remaining in the still [9].The majority of the still-bottom residues were subsequently mixed with waste oils and were used as sprays for the control of dust on roads, parking lots, and horse arenas [9]. One of the horse arenas in east central Missouri was saturated with the waste oil and in the next few weeks, cats, dogs, hundreds of bird and more than 60 horses died. Analyses of the soil found that an estimated 2.8 kg (6.2 lb) of dioxins had been sprayed within the arena [9]. Because the soil of the arena was saturated with the highly contaminated oily and caustic still-bottoms, the animals were directly in contact with the liquid matrix and the exposures were lethal because a toxic dose was received. Removal of the contaminated soil and subsequent incineration were required in the cleanup operations [9].

During the same period, i.e., 1972, similar mixed waste oils and still-bottom residues were sprayed on dirt roads to control dusts in the community of Times Beach, Missouri. During the 1980s, EPA conducted a sampling program of sites throughout Missouri, including the town of Times Beach, where the waste oils and still-bottom residues had been sprayed. EPA reported levels of 4.4-317 ppb in samples taken from the roads [8]. Subsequent studies with Times Beach soils indicated that the 2,3,7,8-TCDD residue that was located a few millimeters below the soil surface was so strongly absorbed by the high organic carbon soil that little upward or downward migration occurred over a 16-month period [10]. The movement of water through the soil profile had little or no effect on the movement of the TCDD. However, it was noted that because of its high lipophilicity, TCDD moved downward in soil profiles in the presence of a dispersing medium such as organic solvents or gasoline [11].

Studies published by the University of Missouri in 1992 concluded that the binding of TCDD to soil approaches irreversibility over time due to the encapsulation of the compound in soil and mineral matter [11]. This and similar observations have led to the conclusion that 2,3,7,8-TCDD probably has a half-life of 25-100 years in subsurface soil, and 9-15 years at the soil surface (i.e., the top 0.1 cm) [7, 12].

The horse arena (noted earlier) and Times Beach were two of 14 confirmed sites in Missouri contaminated by the waste oils and still-bottoms from NEPACCO during 1971-1972 [9]. The cleanup of all sites involved the incineration of almost 2.5 million pounds of dioxin-contaminated materials and was not completed until 1987 [9]. Nine of the sites had soil contamination of 1 ppb or greater, a level at which public health officials concluded that it was reasonable to consider limiting human exposure. This

conclusion was based on the proximity of contaminated soil to humans and concern over potential daily exposure to TCDD by ingestion, skin absorption, or inhalation of contaminated soil [13, 14]. A comprehensive examination in 1986 of 154 exposed and 155 unexposed persons found no excess of clinical illness in the exposed group [13]. A subsequent human reproductive outcomes study in 1988 of residents in the nine sites did not provide evidence that TCDD exposure had a substantial impact on the reproductive outcomes investigated [14]. Yanders concluded:

> *The harm suffered by the majority of Missouri dioxin victims has not been physical illness; there is no increase in clinical illness in the group of exposed people studies, even though the average level of dioxin found in the adipose tissue of residentially-exposed persons is somewhat higher than controls. Their injuries are psychological, social, economic, and the persistent, wrenching belief that their government, which they expected somehow to make things right, has let them down* [9].

Discussion and Conclusion: The Missouri dioxin episode provided an example where improper disposal of industrial wastes resulted in widespread TCDD contamination in the local communities. Direct contact to the TCDD-contaminated waste oils immediately after application resulted in deaths of numerous species of animals. Once the waste oil and TCDD became bound within the soil matrix, its bioavailability decreased significantly. Despite the extent and magnitude of contamination, the impact on the clinical health of the people in the affected community was minimal.

Soil Contamination from Emission Sources

Most studies involving 2,3,7,8-TCDD are studies of sources from industrialized and urbanized areas. These studies involve a number of atmospheric phenomena, including the wet and dry deposition of dioxin-contaminated anthropogenic airborne particulate matter onto soils and vegetation, and the wet and dry deposition of vapor-phase dioxins onto soils and vegetation. Atmospheric deposition is the major pathway for contamination of the terrestrial/agricultural food chain. However, because of wind dispersion and the binding of the dioxin to particulate matter, the concentration of the TCDD on plant and soil surfaces is very low, as is its bioavailability [3, 8, 15]. However, the Seveso, Italy dioxin episode provided a case study where individuals came in to direct contact with liquid droplets containing high concentrations of TCDD [16].

Almost 40 years have elapsed since the Seveso, Italy industrial accident involving a small factory producing trichlorophenol that resulted in the discharge directly into the atmosphere of the contents of a chemical synthesis reactor containing sodium trichlorophenol, sodium hydroxide and approximately 250 g of TCDD. These materials were discharged as a dense white cloud of gases and vapors, liquid droplets, and solid particulates that settled over a residential area on 10 July 1976 [16].

At the time of the accident, children were playing downwind from the factory and were directly in the path of the white cloud of gases and vapors. Within a few days after the accident, the first signs of contamination in the population were skin rashes which affected mainly those children downwind from the chemical release. These skin lesions were burns of the first and second degree caused by contact with the caustic chemicals, primarily concentrated sodium hydroxide, from the cloud and were the early visible indication that the population had been contaminated by the reactor discharge [16]. On 26 July 1976, the Italian authorities evacuated 179 people from a 12-hectare (30 acres) area immediately southeast of the factory (the direction of the cloud) and extending a distance of 730 meters (2,400 feet) from it. A few days later, further findings prompted the evacuation of 557 more people living in the area extending about 2.1 kilometers (1.3 miles) from the plant and covering approximately 73 hectares (180 acres) [16].

Monitoring of the skin complainants was an urgent priority. Over 600 people with skin lesions were referred to a Dermatology Clinic between the end of July and the end of August 1976 [16]. Of the 600 referred, 477 had lesion symptoms primarily associated with the caustic sodium hydroxide, and in which regression and healing occurred within 15-20 days. However, 34 of the 477 were subsequently found to be suffering from chloracne, the hallmark of TCDD exposure, and the majority of these were children [16]. Eventually 42 cases of chloracne were attributed to the Seveso accident with most healing occurring within two years. There were only two or three cases where scarring occurred. There were no deaths attributed to the episode [16].

Following the evacuation of the 73 hectares, the area was fenced off, and access was prevented. This area was designated Zone A where the TCDD levels averaged 235.5 $\mu g/m^2$ (240 ppt in the top 7 cm). Zone B was an area of 220 hectares (544 acres) and was located along the TCDD main distribution pathway, and had TCDD levels that averaged 3.0 $\mu g/m^2$ (or 30 ppt). Zone R (Respect zone or zone of caution) covered an area of 1,200 hectare (~3,000 acres) and had a population of 31,000 and a TCDD level

that averaged 0.5 $\mu g/m^2$ [16]. Immediately after the accident, grass samples in Zone A exceeded 15 ppm TCDD, but after 1 month the levels were < 0.001 ppm. Thus, it was not surprising that domestic animals and some wildlife, particularly rabbits, domestic poultry, and other birds started dying spontaneously within 3 days of the accident. The Italian authorities quickly banned the consumption of vegetables, dairy products and meat from Zones A and B. Final death count of spontaneous deaths exceeded 3,300 animals, but as a prophylactic measure, the authorities slaughtered an additional 78,000 animals [16].

Soil concentrations of TCDD in the top 7 cm of soil dropped rapidly (73%) between August 1976 and December 1976, but decreased very slowing between December 1976 and December 1980 (an additional 8%) [16]. Thus confirming the initial volatility and photodegradation of the TCDD, and the binding of the remaining TCDD to the soil particulate and organic matter.

The burial of the contaminated soil and other materials in a secure landfill was the method selected for the rehabilitation program. The highly contaminated soil (> 1ppb) and waste materials were placed in two basins having a total capacity of 285,000 cubic meters (373,000 cubic yards) of soil. Once filled, the basins were covered with a 1 meter (3.3 feet) layer of soil. Nine years after the Seveso episode began, the work of restoring the community was complete [16].

For more than 25 years, studies of mortality and morbidity have been conducted on the population of Seveso and surrounding communities. No excess of deaths or any particular cause of deaths was noted at five years beyond the accident [16]. At ten years, a mortality study found that incident-related stressors, e.g., cardiovascular causes, were more relevant to increased mortality than TCDD exposure [17]. Long-term studies of resulting health effects confirmed that the main health effect to have been chloracne. Studies also have drawn possible links to neuropathy, liver function, cardiovascular and respiratory diseases, and cancer, but the study results have been conflicting and in some cases and aside from the chloracne have generally been considered to be inconclusive [18, 19].

Discussion and Conclusion: The Seveso, Italy dioxin episode represented the extreme situation where a reactor mixture of sodium trichlorophenol and sodium hydroxide, massively contaminated with TCDD, was aerially discharged as a dense white cloud of gases and vapors, liquid droplets, and solid particulates into a residential community. Those individuals who came

in direct contact with the droplets of sodium hydroxide containing TCDD initially had caustic acid lesions and within weeks were also diagnosed with chloracne, the hallmark of dioxin poisioning. Animals that came into direct and immediate contact with the liquid drops that had dried on the soil surface or on the vegetation that they consumed were also poisoned. However, within weeks researchers determined that the TCDD contained in the soil was not considered the main source of risk. They concluded that as long as it remained in the soil, it could be absorbed virtually only by contact with the skin. The decision, however, was to bury the contaminated soil, thus preventing any future human or animal contact with the dioxin.

Soil Studies Following the Spraying of Agent Orange

The soil residue studies in Missouri and Italy involved soil contamination with TCDD that differed in three major ways from the soil contamination associated with spraying Agent Orange or 2,4,5-T herbicide: 1. The TCDD was present in an alkaline environment, i.e., it was present in a liquid matrix containing caustic sodium hydroxide, and other materials that significantly hindered the photodegradation of the TCDD; 2. The intent of spraying herbicide to impact the vegetation, minimized the amount of soil contamination; and, 3: TCDD photodegration in sunlight was enhanced by the hydrogen donor (as a proton) in the Agent Orange/2,4,5-T matrix, thus reducing the concentration that was bound by the soil [20]. These critical differences were important since testing and subsequent missions involving Agent Orange were conducted in daylight [21].

The primary soil studies of the herbicidal components and the 2,3,7,8-TCDD associated with Agent Orange were the studies conducted at Eglin Air Force Base (AFB), Florida; the former Herbicide Storage Sites at the Naval Construction Battalion Center (NCBC), Gulfport, Mississippi, and Johnston Island, Central Pacific Ocean; and in Vietnam. The studies conducted in Vietnam were those conducted by the National Research Council of the National Academy of Sciences in 1971 and 1972, and the studies conducted decades after the conflict ceased. Those studies conducted in the late 1990s and early 2000s provided evidence of TCDD presence primarily in soils associated with RANCH HAND or US Army Chemical Corps military operations at former storage and loading sites.

Soil Studies Conducted in Vietnam

The last mission involving Agent Orange was conducted in Operation RANCH HAND in April 1970 [21]. Teams of scientists selected by and representing the National Research Council of the National Academy of Sciences visited Bien Hoa Province in South Vietnam between September 1971 – August 1972, and collected soil samples in the mangrove areas of Vung-Tau and Rung Sat, and in forestry areas near Ban-Me-Thuot [22, 23]. Sites were selected at locations where a history of spraying Agent Orange was available. Only the herbicides 2,4,5-T and picloram (from Agent White) were detected in most samples and then primarily in the top layer of the soils at concentrations ranging from 3 ppb to 3 ppm of 2,4,5-T, and less than 0.4 ppm picloram [22]. One of the soil samples collected in October 1971 from a site that had received repeated applications of Agent Orange in the period 1965-1970, had a soil level of 0.01 ppm 2,4,5-T, but no analysis of TCDD were conducted on any of the samples because of the lack to analytical capability to detect it. The soil samples were subsequently treated in the laboratory (uniformly mixed) with sufficient Agent Orange to result in a 2,4,5-T concentration of 15 ppm, and were incubated for 160 days. More than 90% of the 2,4,5-T disappeared within 80 days. The authors concluded that these Vietnamese soils were inherently capable of degrading 2,4,5-T at levels at least as high as15 ppm [23]. The National Research Council concluded from their studies in Vietnam:

> *Claims that the herbicides as they were used during the war have rendered the soil "sterile," permanently or at least for prolonged periods, are without any foundation. It should be noted that these claims were contrary to all existing information for the herbicides in question* [22].

Schecter et al (2001) reported soil levels of 0.6 to 1.2 ppm TCDD at the site of a former Agent Orange spill of 5,000 gallons in 1970 at Bien Hoa Airbase in Southern Vietnam [24]. However, no protocol information was provided on date, how the sample was collected, or to what depth. Sediment samples collected (again without describing the sampling protocol) from Lake Bien Hung (described as "close" to the former airbase) and from sites in and around Bien Hoa City had levels of TCDD from non-detected to 177 parts per trillion. Including 2,3,7,8-TCDD, all of the samples contained detectable levels of 17 different dioxin and furan congeners. This subsequent observation led Mae to challenge these data noting that waste waters and emissions from the Bien Hoa industrial zones have been routinely

discharged directing toward and into waters feeding Lake Bien Hung [25]. The waste waters contained wastes from paper, plastic, electric, and chemical industries, all sources of dioxins and furans [25]. These observations of such sources were similar to those for sediment studies in the United States from the Saginaw River and Bay and from Lake Huron [26], or in China for the lower reaches of the Yangtze River [27].

Dwernychuk et al (2002) detected 2,3,7,8-TCDD in soils collected from a former Special Forces base in the Aluoi Valley of central Vietnam where Agent Purple and Agent Orange were stored and sprayed on the exterior of the base perimeter prior to December 1965 [28]. Levels of TCDD detected in the top 10 cm (collected from 1996 – 1999) of soils or sediments ranged from 1.8 to approximately 900 ppt (0.9 ppb). The highest levels were attributed to "hot spots" where the herbicide had been spilled. Additional studies of "hot spots" have been conducted on sites where tactical herbicides were stored and loaded on RANCH HAND aircraft or helicopters of the US Army Chemical Corps [29].

Studies Conducted at Eglin AFB, Florida

From 1961 – 1971, the Air Development Test Center, Eglin AFB, Florida developed, tested, and calibrated the aerial spray systems used in support of Operation RANCH HAND and the US Army Chemical Corps in Vietnam. Twenty major test and evaluation projects of aerial spray equipment were conducted on four fully instrumented test grids, each uniquely arrayed to match the needs of fixed-wing, helicopter, or jet aircraft. Each of the grids was established with the boundary of Test Area C-52A of the Eglin AFB Reservation [30]. The tests, conducted under climatic and environmental conditions similar to those in Vietnam, included the use of the herbicides Agent Orange and Agent Purple. Approximately 165,400 lb of 2,4,5-T and 167,600 lb of 2,4-D were aerially disseminated on an area of less than 1 square mile. Data from the analysis of archived samples suggested that an estimated 6.8 lb of 2,3,7,8-TCDD, present as a contaminant, were aerially released in the test area [30].

In 1974, studies were initiated on the soil persistence and movement of 2,3,7,8-TCDD. The oldest grid on the Test Area, Grid 1, was used from 1962 – 1964, and received 71,440 lb of 2,4,5-T from Agent Purple. It was estimated that on an area of less than 91 acres approximately 6.4 lb TCDD was aerially disseminated with the herbicide. The aerial distribution of herbicides on the test grid was neither uniform nor random, but rather along

discrete sampling arrays to measure particle size and deposition. Thus, by considering the flight paths, water sources, and terracing effects, 22 soil samples were collected from the top 15 cm of soil from 1974 through 1978 and analyzed for 2,3,7,8-TCDD. The levels of TCDD varied from less than 10 ppt to 1,500 ppt (1.5 ppb) with a median of 110 ppt and a mean of 325 ppt [30, 31].

The selection of a 15-cm (6-inch) soil core profile was based on studies that indicated that TCDD concentrations in the 0-2.5 cm ranged from 150 to 460 ppt; 2.5-5.0 cm level ranged from 160 to 815 ppt; 5.0-10.0 cm level ranged from 700 to 2,400 ppt; and the 10.0-15.0 cm level range from 44 to 1,100 ppt. Essentially, no TCDD was detected below 15 cm. Although the levels of TCDD were greatest in the 5.0-10.0 cm zone of the soil profile, it was concluded that it was unlikely that these data represented leaching of TCDD though the soil profile [31]. Rather, a more likely explanation was that the TCDD was deposited in layers, during and in subsequent years after herbicide application, as a consequence of wind and water movement of the contaminated soil particles. Examination of the soil horizons in excavated profiles of Grid 1 clearly showed that within the top 15 cm discrete layers could be discerned that differed from the parent soil [31]. In reviewing climatic data including wind speed and direction, it was noted that the winds that occurred in the evenings after the herbicide was aerially disseminated resulted in contaminated soil particles being moved back and forth across Grid 1, and eventually being deposited in low areas of Grid 1. It was likely that water also moved the contaminated particles into the low-lying areas of Grid 1 [31]. Similar observations of the soils and TCDD contamination of Zone A, Seveso, Italy were made [32]. In both studies, it was apparent that TCDD was very persistent once it was bound within the soil profile.

It was calculated that 87% of the TCDD in the herbicide applied to the Test Area impacted the 91-acre Grid 1. Approximately 6.4 lbs of TCDD needed to be accounted for on Grid 1. If the mean value of 325 ppt was used as the level of TCDD in 1978 for the top 15 cm of soil and the density of the Lakeland Sand was 1.4 g/cm^2, then approximately 1 percent of the TCDD remained 14 years after application. Most of the TCDD in Agent Purple (and Orange), when exposed to natural sunlight at each of the times of application, was likely lost to photochemical degradation [30, 31].

Studies Conducted at the NCBC and Johnston Island

At the conclusion of the Vietnam War, the United States Air Force had more than 15,000 55-gallon drums of Agent Orange that was not shipped to Vietnam, but rather put in storage at the Naval Construction Battalion Center (NCBC), at Gulfport, MS [33]. In addition, in Project PACER IVY in March 1972, the US returned more than 25,000 drums of Agent Orange from Vietnam to Johnston Island, Central Pacific Ocean [33]. Both inventories of herbicides were destroyed by at-sea incineration in 1977, and a soil residue monitoring program was initiated at both locations in 1978.

The soil level of herbicides (2,4-D and 2,4,5-T) at spill sites dramatically decreased at both NCBC and Johnston Island from a maximum of 62,000 mg/kg (ppm) (8 samples taken from the top 10 cm (~4 inches) of soils from spill sites from each of the two former storage sites) to less than 2% of the initial concentration remaining at the end of the 4 years (1978 – 1982). In the same sampling period and sample sites, the TCDD concentrations decreased from 180 ng/kg (ppb) to less than 100 ppb (45% loss in 4 years) [34]. During the remediation program for NCBC in 1986, 35 soil cores were collected on the former Agent Orange Storage Site at NCBC. Essentially no herbicide was detected; however, in the soil profile increment of 0-8 cm (~3.1 inches) TCDD levels ranged from <0.01 – 310 parts per billion (ppb) (generally in the same range that was detected in 1982). For the 8-16 cm increment the TCDD levels ranged from <0.01 – 93 ppb; and, for the 16-24 cm increment, the TCDD levels from <0.01 – 12 ppb. No TCDD was detected below 24 cm (~ 10 inches), with a detection limit of <0.01 ppb [35]. The movement of the TCDD in the soil profile was thought to be associated with the mass movement of liquid Agent Orange into the profile. Similar results were obtained for Johnston Island [35].

Discussion and Conclusion: In the aerial spraying of tropical vegetation or dense shrubs with tactical herbicides, the intent was to spray and thus control the vegetation. Studies conducted by USDA in Puerto Rico and Texas indicated that the vegetation intercepted 94% of herbicide while only 6% landed on the soil beneath the vegetation [33]. Thus, soil bound TCDD levels from a area that had been repeatedly aerially sprayed with Agent Orange in Vietnam ranged from 1 to 41 ppt (average 8.8 ppt) [36]. However, TCDD concentrations in soils where the Agent Orange was spilled contained concentrations from 0.6 to 1.2 ppm (orders of magnitude greater than from aerial applications). However, in either case, once the TCDD came into contact with the soil, it was rapidly and tightly bound. In the Eglin Studies,

99% of the TCDD was photodegraded within hours of its aerial deposition on to the bare sands of the test area. Remaining 1% was bound within the top 6 inches of soil due to wind and water movement, where it persistent for at least two decades. In the studies of NCBC and Johnson Island Agent Orange storage sites, the herbicides rapidly degraded primarily by microbes, but TCDD was much more persistent requiring incineration of the soils 20 years later [35].

UPTAKE OF TCDD FROM SOILS AND SEDIMENTS

Test Area C-52 at Eglin AFB offered a unique opportunity to study the impact of soil-bound TCDD in a terrestrial ecosystem. Most of the vegetation had been removed in 1961 before establishing the four fully instrumented test grids used in support of the aerial calibration tests and evaluations of aerial spray equipment subsequently used in Operation RANCH HAND and by the US Army Chemical Corps in Vietnam [30, 31, 33]. Because of the importance of the calibration, the decision was made to use the actual tactical herbicides that would be used in Vietnam, namely, Agents Purple, Orange, White and Blue. The removal of the vegetation, and hence high solar exposures, provided an opportunity to follow ground-based residues independent of canopy interception [30].

Studies of the soils, fauna, flora, and aquatic ecosystems of the test grids and associated perimeters were initiated in 1969 and concluded in 1984 [31, 30]. More than 340 species of organisms were observed and identified within a 1.5 square mile area encompassing the four test grids and their perimeters. More than 300 biological samples were analyzed for TCDD and detectable residues were found in 16 of 45 species that had been collected and carefully examined for any anomalies [30]. An examination of the ecological niches of the species contaminated with TCDD residues confirmed that each was in close contact with contaminated soil. Anatomical, histological, and ultrastructural examinations, spanning more than 50 generations of the Beachmouse, *Peromyscus polionotus*, the dominant rodent on the test area, demonstrated that continual exposure to soil concentrations of 0.1 to 1.5 parts-per-billion of TCDD had minimal effects upon the health and reproduction of this species [30].

The aquatic studies at Eglin were consistent with the literature review of polychlorinated dibenzo-p-dioxin and dibenzofurans in the aquatic environment conducted by AEA Technology in England [37]. They concluded that the dominant transport mechanism for removal of 2,3,7,8-

TCDD from water is by sedimentation of soil and organic particles, although some volatilization will occur. Sediment re-suspension and remobilization of the TCDD will vary on a site-by-site basis depending on the nature and extent of physical processes (e.g., winds/ waves/currents) and biological processes (disturbance by benthic organisms) [37]. Aquatic organisms can bioaccumulate the TCDD by ingesting contaminated soil particles, although it was concluded that the total quantity (mass) of TCDD in the biota in a given water body will account for only a small fraction of the total quantity of TCDD in that water body [37].

The University of Missouri-Columbia initiated a study of the terrestrial ecosystem associated with the abandoned town of Times Beach, an area similar to the test grids at Eglin AFB [38]. The Deer Mouse, White-footed Mouse, and the Prairie Vole, all rodents nesting in contaminated soil had concentrations similar to those found in the Beachmouse at Eglin AFB, 47 – 1,736 ppt, whole bodies and livers, respectively [30, 38]. The Missouri scientists made additional comparisons with Seveso animal studies and found similar soil and tissue results. Having an understanding of the levels of TCDD in the body fat of animals, the authors then made a comparison to available monitoring studies (as of 1987) of the adipose tissue of man [38]. Their observations were as follows:

> *The concentrations in over 500 human adipose samples containing TCDD, which range from <1 to 1,840 ppt and with a mean of 79.6 ppt, can be divided into four groups. In the first group, the means of approximately 277 samples of low-exposure individuals from a variety of locations in the US, Canada, Sweden, Germany, Japan and Vietnam was 6.7 ppt (range 1.4-12.7 ppt). In the second group of moderate exposure, the means was 16.9 ppt (range 3.7-41.1 ppt) in 84 individuals exposed to TCDD in Southern Vietnam (Vietnamese and US Army personnel), the Binghamton, New York Post Office fire, and personnel in the non-production area of a facility manufacturing TCDD contaminated chemicals. A third group of 206 individuals subjected to high occupational and recreational exposure had TCDD levels of 3.5 to 978 ppt and a means of 193 ppt. The fourth and last group of the highest exposure was the woman from Seveso who had a level of 1,840 ppt* [38].

It is likely that most of the 500 human samples did not represent TCDD obtained through working with contaminated soils, but rather through ingestion of contaminated food, e.g., meat, fowl, and fish [24, 25, 29].

The IOM in describing how Vietnam veterans might have been contaminated with TCDD suggested: "*troops who moved through defoliated areas soon after treatment may have been exposed through soil contact, drinking water, or bathing* [1]". However, the physical properties of TCDD (essentially insoluble in water) likely made drinking water or bathing a negligible route of contamination, but could contact with the soil be a viable route of contamination? Numerous studies of dermal contact with TCDD have found that any exposures to contaminated soil that would have occurred were "negligible" because the skin is a major barrier to TCDD uptake, contributing less than 1% over the long-term to the body burden [39]. A validation of this observation occurred with some of the field researchers who were involved in the ecological studies at Eglin AFB, Florida [40].

Beginning in 1970 (before any analytical results of TCDD on the Eglin AFB Test Range C-52A in 1974), three men collected thousands of soil samples bare-handed and in direct contact with the soil [40]. These individuals were also involved in conducting soil bioassays and in the capture of contaminated animals with contaminated pelts. Two of three also participated in the cleaning of the ship, the *MS Vulcanus*, after the incineration of Agent Orange in 1977, where metal scrapings were contaminated with TCDD. In April 1979, all three individuals volunteered for abdominal fat biopsies; these samples were analyzed for TCDD. The results of those analyses varied from 5 to 7 parts-per-trillion (ppt); the normal background level at that time being 3-4 ppt [40].

CONCLUSIONS

From a review of the available data it was concluded that when 2,3,7,8-TCDD entered the environment, it was rapidly bound to soil and organic particles. Its low water solubility and low vapor pressure resulted in its failure to move in the soil profile, while at the same time these properties enhanced its long-term persistence. In water sources contaminated with TCDD, the residue was found to be bound to the soil particles that comprised the soil sediment; certain aquatic species were contaminated from ingesting the sediment. Animals that came into contact with a liquid matrix

containing the TCDD were likely poisoned. However, animals that came into contact with "aged" contaminated soil could become contaminated, but the level of contamination was generally insufficient to have adverse effects upon the animals. In humans, handling contaminated soil resulted in negligible contamination since the skin acted as an effective barrier to the uptake of the TCDD.

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